

Community and Occupational Studies of Lung Cancer and Polycyclic Organic Matter

by Jorma Rantanen*

Finland is used as a model in attempts to study the possible association of the incidence of lung cancer and exposure of population to fossil fuel combustion products. Unfortunately because of great geographical variation of unknown origin in the incidence of lung cancer in Finland, detailed studies of the possible role of an individual exposure in the lung cancer risk are not possible. This background variation in the incidence is much greater than variation carried by any known etiological factor and does not clearly correlate with the degree of urbanization, industrialization, regional use of fossil fuels, number of motor vehicles or smoking habits.

To get more precise information on the possible association of lung cancer incidence with exposure to fossil fuel combustion products, occupational studies serve as powerful tools. The definition of population is more reliable and the measurement of exposures can be done more precisely; moreover the management of confounding and modifying factors is more effective than in community studies. So far the studies carried out among the Finnish working population exposed to PAH compounds reveal an association between the lung cancer risk and exposure to PAHs.

Introduction

So far most of the increased cancer risks and of the urban-rural difference in lung cancer rates have been attributed to smoking. Occupational exposures and life-style factors other than smoking are thought to play minor roles in the lung cancer risk in industrialized countries. The combustion products of fossil fuels in industry and in heating and the exhaust products of motor vehicles have been intensively studied for their possible role in the epidemiology of cancer among urban populations. To date the overwhelming impact of smoking has made it difficult to find associations between cancer rates and the combustion of fuel. The detection of the possible contribution of fuels, however, may be important for the prevention of cancer among both smokers and non-smokers residing in urban areas.

Another approach to research into environmen-

tally determined lung cancer is to study occupationally exposed populations, even though exposed groups may comprise only a small proportion of the total population. Both exposure and outcome can be more precisely recorded in occupational settings than in studies of general populations. Also, in most cases, the exposure levels are of higher magnitude than in the urban air; therefore a clearer effect can be found among the occupationally exposed than among the general population. In the present paper Finnish studies on lung cancer are reviewed as a background for discussions of difficulties in the studies of environmentally determined cancer.

Community Studies

Urban-rural differences in the incidence rates for lung cancer have been well documented in several industrialized counties (1), but not all of the causative factors contributing to this difference have been detected. As early as 1955, Stocks and Campbell (2) discussed an absolute urban excess in a

*Institute of Occupational Health, Haartmaninkatu 1, 00290 Helsinki 29, Finland.

Liverpool population stratified according to smoking habits. About 50% of the deaths from lung cancer in Liverpool were attributed to smoking, and approximately 37% to a factor almost completely absent in rural areas. This estimate corresponded well with the values of BaP in Liverpool, which were about ten times higher than in rural areas. Several reports on urban-rural differences have analyzed various causative factors since this early study, but little evidence on a specific contributor has yet been found (3-5).

In Finland the incidence rates for lung cancer have been among the highest in the world ($77.8/10^5$ for men and $5.5/10^5$ for women in 1977). The rates for men are twice as high as in Denmark and three times as high as in Sweden and Norway (6). The incidence rates have been declining for the last five years. In 1940-1969, smoking was as prevalent among Finnish males as it was in those industrialized countries with highest smoking rates, whereas during the 1970s, smoking was less prevalent in Finland than on average in the industrialized world (7).

A clear urban-rural difference can be seen in the Finnish cancer statistics, but the difference is currently decreasing for men whereas it is increasing for women (Table 1). The decline in the total incidence of lung cancer observed in recent years is due to the decline in the risk of urban men (6).

There is considerable regional variation in the risk for lung cancer in men, which ranges from $59.2/10^5$ in Åland to $103.0/10^5$ in North Carelia. In general there is a clear difference between cancer rates in the southwest ($59.2-80.2/10^5$) and the northeast ($81.7-103.0/10^5$) (Fig. 1). Even the provinces with the lowest incidence rates have age-adjusted incidence figures per 100,000 population which are twice as high as those of New York or any other American urban area (8,9). Especially striking is the high incidence rate of lung cancer in North Carelia ($103.0/10^5$), where the incidence rate exceeds the highest figures for industrial areas in the U.S. (9). North Carelia, however, is comparatively sparsely populated; in wide areas of the province, less than 30% of the population is employed in industry.

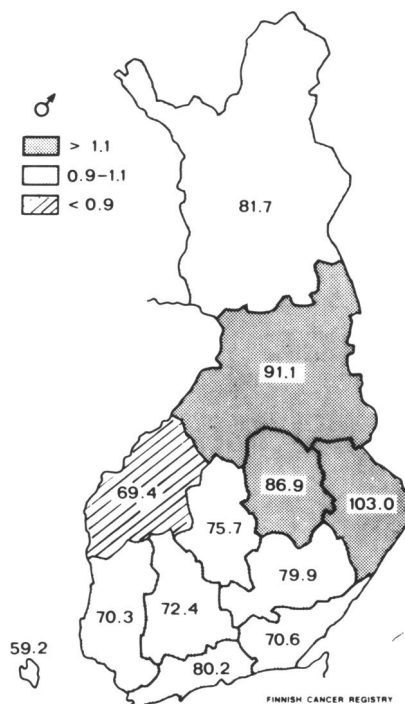


FIGURE 1. Age-adjusted average morbidity in lung cancer among Finnish males in various provinces, 1970-1974. Whole country average, $77.5/10^5$. The stratifications indicate relative rates (whole country-100) (10).

Interestingly, the risk of coronary heart disease is highest for the same areas with the highest incidence of lung cancer. The excess risk in North Carelia cannot be explained by industrialization, and even less by air pollution. Smoking was more prevalent in the 1950s and the 1960s in the northern and eastern provinces, but since the 1970s no statistically significant difference can be found (7) between different parts of the country.

The local incidence rates of cancer (including respiratory cancer) was very recently studied (10). The risks of cancer were found to correlate with the municipal status of 20 years ago, with the degree to which the environment was built up, with social class, with the degree of industrialization and with

Table 1. Age-adjusted incidence rates (per 100,000 person-years) of cancers of the nose, larynx and lung in 1955-1974 in the three biggest towns (Helsinki, Tampere, Turku), other towns and rural areas of Finland, by sex.^a

Area	Nose		Larynx		Lung	
	Males	Females	Males	Females	Males	Females
Biggest towns	1.3	0.6	9.5	0.6	97.0	8.1
Other towns	1.3	0.5	8.2	0.4	89.7	5.2
Rural areas	1.1	0.7	6.4	0.3	75.1	4.3

^aData of Teppo et al. (10).

several other parameters. In spite of a clear urban-rural difference in the incidence rates for lung cancer, on average, the rural municipalities of eastern Finland had the highest risk rates.

The use of liquid petroleum products ("heavy" industrial oil, heating oil, diesel oil and gasoline) was not found to correlate with incidences of respiratory cancer (11). On the other hand, the combustion of solid fuels was found to contribute to the risk. In Finland, the use of coal, which is limited to a few urban areas in the south and the southwest (Helsinki, Turku), comprised 3.128 tons in 1960 and 3.579 tons in 1975. In other parts of the country, the use is minimal. The annual consumption of liquid petroleum products is about 12 million tons, i.e., an average per capita consumption of about 2.5 tons. At a national level, the annual incidence of lung cancer per 100,000 population correlates well with the annual consumption of liquid petroleum products (Fig. 2) between 1960 and 1977, whereas no correlation was found between the cancer incidence of the provinces and the provincial total or per capita liquid fuel consumption in 1977. Nor did the provincial incidences of lung cancer correlate with the number of motor vehicles in provinces.

So far no epidemiological study on the incidence of lung cancer and air pollution has been done in Finland. On the basis of our knowledge about the incidences of cancer in municipalities, however, one can expect that geographical variation in the risk of lung cancer is not caused by ambient air pollution. The excess risk in the municipalities of the eastern provinces is higher than the excess which could be due to smoking or air pollution. Some factor or

factors in the eastern provinces increase the rates to about 30 cases/10⁵ above the rates for typical industrial areas in western Finland.

There have been only a few Finnish studies of the concentration of combustion products of fossil fuels in ambient air. In Helsinki, the concentrations of BaP in the city air range from 0.5 to 5 ng/m³; the background level in rural areas is not known. Due to the high aromatic content of petroleum fuels, the concentrations of benzene in the ambient air may be surprisingly high. Häsänen et al. (12) measured the mean aromatic emissions of 31 cars which underwent a total of 107 ECE 15 tests drives. They found a mean emission of 250 ± 100 mg/km for benzene; the other figures were 350 ± 130 for toluene, 220 ± 95 for *m*-xylene, and 90 ± 5 ± 40 for *o*-xylene. Of the aromatic compounds in the fuel, 3-4% were expelled unburned, with the exhaust products. The concentrations of aromatic hydrocarbons in the city air during rush hour were 100 µg/m³ for benzene, 140 µg/m³ for toluene, 60 µg/m³ for *m*-xylene and 30 µg/m³ for *o*-xylene.

Hammond (13) has described the difficulties in community studies of cancer and air pollution. Precise recording of exposures, both qualitatively and quantitatively, causes great difficulties. The mobility of subjects within the country and within a city or town and their daily mobility between different parts of urban areas make it difficult to measure the actual exposures. In addition, great seasonal weekly and daily variation occurs in both the concentrations and the composition of air pollutants such as exhaust products. Therefore it is difficult to get an accurate picture of the prevalence of pollutants in large areas of cities. Furthermore individual exposure to the pollutants present in the city air varies according to a number of factors which cannot be controlled. Finally food, drinking water, and ambient air involve very many other uncontrolled confounding or effect-modifying factors, and the use of alcohol, smoking, and occupational exposures must also be considered (Table 2). All these interfering or contributing factors should be controlled before any conclusions on the specific health effects of air pollutants can be made. For instance, Finns' total exposure to toxic chemicals from all sources (in their food, water, and environment) varies between 1265.6 and 12753.4 mg/day (14). On the other hand, inaccuracy in measurement of exposure, caused by statistical reasons (regardless of the direction of the inaccuracy), always means some dilution of effect, which thus masks the effects in cases where moderate or high exposure levels are studied.

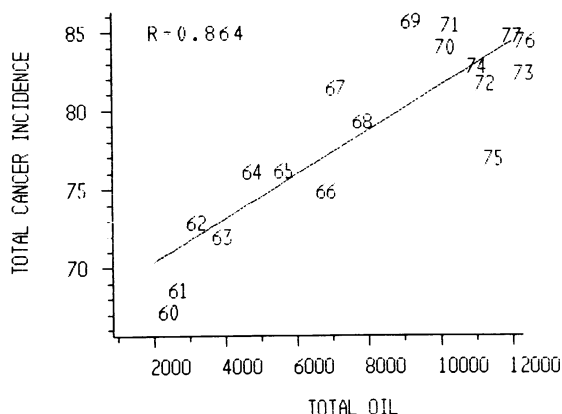


FIGURE 2. Correlation of lung cancer incidence with oil consumption in Finland in 1960-1977. The abscissa shows national consumption of oil products (gasoline, diesel oil, industrial oils); the ordinate shows lung cancer incidence (both sexes) as cases/10⁵.

Table 2. Daily exposures of Finns to hazardous chemicals.^a

Source	Compound	Dose, µg/day
Food		
Metals	As	60
	Hg	6
	Cd	13
	Cr	30
	Pb	70
	Ni	130
	Se	30
Mycotoxins	Aflatoxin	< 0.01
	Fusarium	?
	Ocratoxin	?
	Patulin	10
	Penicillaminic acid	?
	Sterigmatocystin	?
Organic pollutants	PaH	20
	Chlorinated biphenyls	5
	Chlorinated phenols	< 1
Natural compounds	Mutagenic flavonoids	50000
	Hydrazines	1
	Coumarin	?
	Nitrates	14000
	p-Sorbinic acid	?
	Safrols	?
	Tannins	150000
Food additives	Butylated hydroxyanisole	}
	Butylated hydroxytoluene	
	Carragenin	?
	Nitrates	6000
	Nitrite	7000
	o-Phenylphenol	?
	Chloroprophame	?
	Propame	?
	Technatsene	?
	Saccharin	9000
	Sorbic acid	45000
	Cyclamate	40000
	Synthetic dyes	2000
Pesticide residues	130 compounds	150
Substances produced in cooking	Amino acid derivatives	
	Glu	0.1
	Try	0.1
	Nitroso compounds	5
	PaH	20
Drinking water	Chloroform	200
Room air	Formaldehyde	2000
Ambient air	Asbestos	0.1
	Benzene	10
	PaH	0.1
	Metals: Cd	0.01
	Pb	10
Alcohol	Acetaldehyde	10000000
Cigarettes	Acetaldehyde	2000
	Benzene	200
	Formaldehyde	50
	Cd	1-4
	Nitroso compounds	1
	PaH	1

^aData of Hietanen and Hirn (14).

Occupational Studies

Because air pollutants and numerous other environmental factors are found in higher concentrations both in the work environment and also indoors

in well-defined areas, occupational studies provide a number of possibilities to avoid the difficulties in the registration of exposures and in the control of confounders.

A rough analysis of the national mortality data

may already provide some clues about the possible associations between lung cancer and environmental factors. In Finland, Sauli (15) found overmortality from lung cancer among ten high-risk occupations involving heavy industrial work and low socioeconomic status (SMR 1.53-2.25). On the other hand, low risks were found for white collar occupations and certain outdoor workers occupations (SMR 0.45-0.69).

Finnish miners were found to have an increased risk of lung cancer. The SMR of miners was 2.08, compared with 1.0 for the entire economically active population (16) (Table 3). Several types of risks concentrate within the population of miners, including high mortality from accidents and cancer (15). In Sweden, risk of lung cancer for miners was five times as high as among the Swedish on average. Hence the magnitude of absolute risk is of the same order for Swedish and Finnish mines (17), even though the ores mined are completely different.

The emission of radon was found to be the causative factor underlying the increased risk to Swedish miners in mines where exposure to exhaust products was not possible. Radiation may also play a role in Finnish miners increased risk, but the exhaust products of diesel engines, which came into use in the 1950's and the early 1960's, may also play

a role. For this reason, the PaH levels of the ambient air were studied in Finnish mines (18). The total concentration of particulates varied between 0.9 and 27 mg/m³, and the total concentration of PaH ranged between 181 and 801 ng/m³ (Table 4). As indicated in Table 4, BaP was a poor indicator of the total concentration of PaH in the mines.

The possible role of exposure to diesel engine exhaust fumes in the elevated risk to lung cancer of miners is currently being studied in Finnish mines. Because the excess risk has already been found for various mining occupations, future studies will be directed toward measurement of the exposures as precise and as detailed as possible. This will be done both by personal monitoring of the ambient air and by personal biological monitoring of mutagenic activity in urine.

To enable the effects of polycyclic organic matter to be studied further, the exposure of various occupational groups to PaH was measured in Finnish foundries (19) (Table 5). The concentrations of BaP measured in the foundry air were compared with the results of Salmonella TA 98 + S9 mix and TA 100 + S9 mix tests. Strong correlations were found (Fig. 3). So that personal exposure and the possible role of exposures via other routes than the lungs could be measured, the mutagenicity of foundry workers' urine was measured (Table 6) (20). The results of the biological monitoring agreed well with the data obtained from monitoring of environmental exposures.

The concentrations of PaH found in factories with the highest exposure levels varied between 1.0-1.9 µg/m³ in the air and 0.2-1 µg/m³ in the dust. The mutagenicity of the urine of workers employed in these works was also the highest; it ranged from > 58 to 3.630 revertants/1000 mL urine for molders and 1.110-2.860 revertants/1000 mL for casters. The great variation was apparently due to smoking, but even the values for nonsmokers varied between 500 to 1.000 revertants/1000 mL of urine.

Table 3. Incidence of lung cancer in men aged 35-69 in mining and quarrying.^a

	Lung cancer incidence per 10 ⁵		
	Observed	Expected	SMR
Whole branch	42	20.2	2.08 ^b
Mining	27	13.0	2.08 ^b
Deep drilling	2	1.4	1.42
Concentration	4	1.4	2.92
Other mining and quarrying	9	4.4	2.03

^aData of Pukkala et al. (16).

^bStatistically significant at level $p \leq 0.001$.

Table 4. Concentration of particulates, total PAH and PaB in the mine atmosphere.^a

Sample	Particulates, mg/m ³	Total PAH, ng/m ³	BaP, ng/m ³	BaP/total PAH, %
1	2.0	408	17	4.2
2	0.9	226	9	4.0
3	2.5	181	7	3.9
4	2.7	470	18	3.8
5	1.0	244	8	3.3
6	4.0	581	21	3.6
7	7.7	443	13	2.9
8	6.2	538	21	3.9
9	27	426	6	1.4
10	19	673	36	5.3
11	27	801	12	1.5
12	13	569	18	3.2

^aData of Hakala et al. (18).

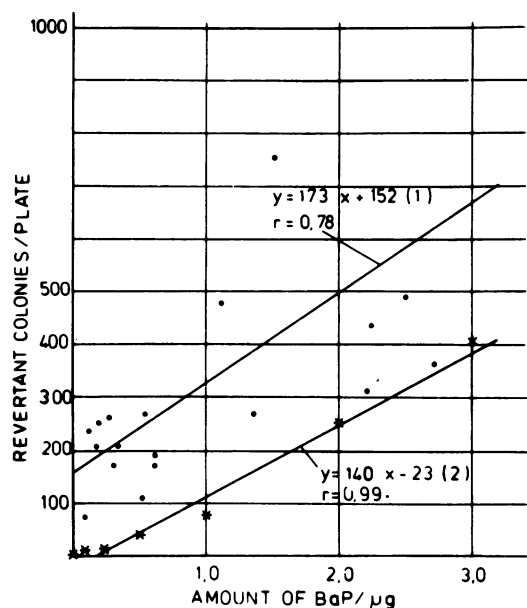


FIGURE 3. Correlations between the amount of BaP and the mutagenicity in foundry A: (1) samples; (2) BaP standards. Mutagenicity determined with TA 98 + S-9 mix (20).

Several studies on lung cancer in various types of foundries (21) indicate clearly increased cancer rates due to occupational exposures. The Finnish foundry study (22) comprised 3876 men. Their total mortality was lower than expected on the basis of the figures from the national reference population, but the mortality from lung cancer was clearly elevated (SMR 1.50). The excess rates for lung cancer centered on iron foundries, where the SMR for the highest risk groups was 2.70. The iron foundry workers were studied with an ambidirectional case-control/cohort approach (23). There were 51 cases of lung cancer, of which 57% were in the group of workers heavily exposed to compounds with PaH, whereas the respective figure for the controls was 43%. The risk ratio between heavy vs. low exposure to BaP was 1.7. Though the difference between various exposed groups (heavy vs. low exposure) was not statistically significant, there was a statistically significant increase in the total number of cases of lung cancer (51.0) among the entire group of iron foundry workers when compared with the expected value (35.3). The increased risk was found among those workers who carry out the particular jobs which involved exposure to high

Table 5. Level of benzo(a)pyrene in the atmosphere of iron foundries during different work phases.^a

Work phase	No. of samples	BaP concentration in air, µg/m ³		BaP concentration in dust, µg/mg	
		Mean	Median	Mean	Median
Melting	18	0.2	0.05	0.04	0.02
Molding	16	1.9	0.1	0.2	0.02
Casting	50	1.0	0.3	1.0	0.3
Shake-out	42	4.9	0.1	0.4	0.05
Pettling	7	0.2	0.2	0.03	0.01
Sand preparation	1	72	72	0.26	0.26
Coremaking	1	0.2	0.2	0.02	0.02
Transport and others	10	0.9	0.6	0.2	0.2
Total	145	2.5	0.1	0.5	0.06

^aData of Schimberg et al. (19).

Table 6. Mutagenicity of the urine of various occupational groups in an iron foundry.^a

Worker ^b		Total dust, mg/m ³	BaP, µg/m ³	Mutagenicity, rev/1000 mL
A(S)	Molding	3.0	0.07	2050
B(S)	Core making	6.1	0.19	> 1430
C	"	4.2	0.11	920
D(S)	"	5.8	0.22	3630
E(S)	"	5.1	0.10	> 580
F(S)	Fettling	16.2	1.08	1110
G(S)	Casting	2.4	0.12	> 2360
H	Pour out	14.7	0.26	2050
I	Preparing	4.1	0.13	> 1390
J(S)	Machine handling	2.2	0.10	> 2370
K(S)	"	2.8	1.01	1190
L	Laborer	5.6	0.14	910

^aData of Schimberg et al. (19).

^b(S) = smoker.

concentrations of PaH, and the urine of these workers had the highest mutagenicity values. Smoking and other possible confounding and modifying factors were carefully controlled. Although conclusions should be made with caution, one can suggest that polycyclic aromatic hydrocarbons in the air at the foundry are responsible for the increased risk of lung cancer.

Compared with community studies, occupational approaches provide certain advantages: the populations are easily defined, and their follow-up is possible by using company personnel records. On the other hand, it is possible to record precisely the level of exposure in the workplace air and in the workers' breathing zones. Personal biological monitoring of, e.g., chromosomal changes or mutagenicity in the urine can be carried out.

Chromosomal aberrations have been reported to increase as a result of exposure to benzene (24,25). Swedish workers handling motor fuels (e.g., road tanker drivers) and industrial workers exposed to benzene had increased frequencies of aberrations, but this was not the case with either ship tanker crews or the staff of the filling stations. However,

miners operating diesel engines did not have increased rates of aberrations when compared with office employees and construction workers (26). The exposures to diesel fuel were concluded to be so low that the doses required to yield aberrations are not achieved.

The biological monitoring of workers by measuring the mutagenicity of their urine by fluctuation tests (27) has been carried out in several types of occupations. Besides foundry workers, these occupations include nurses handling cytostatic drugs, rubber workers, and workers exposed to various industrial chemicals. Parallel studies have been done to measure the frequencies of chromosomal aberrations and sister chromatid exchanges in exposed workers. By detailed recording of exposures (with the help of personal hygienic and biological monitoring) and by combining cytogenetic studies to these approaches, epidemiological studies of even small occupational groups with comparatively high levels of exposure may provide much more information on the risks of the combustion products of fossil fuel than is provided even by extensive community and population studies which encounter all the methodological difficulties listed above.

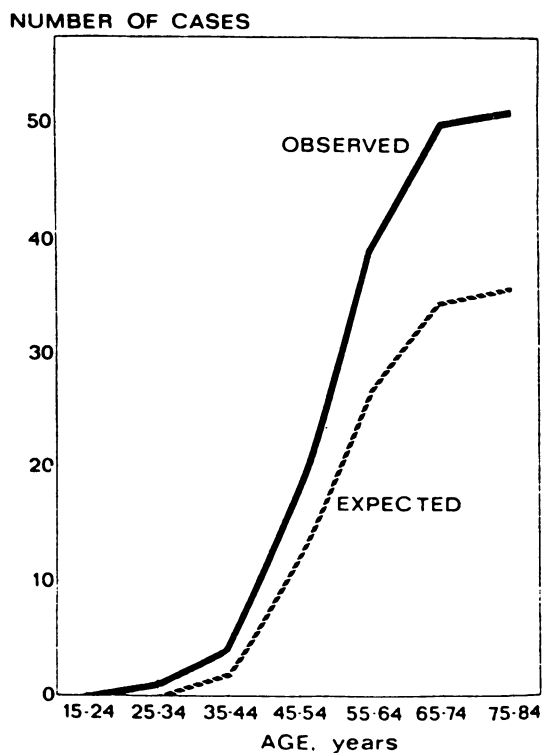


FIGURE 4. Observed and expected cases of lung cancer among Finnish male iron foundry workers. The expected values are based on the proportional mortality figures for the Finnish male population (23).

REFERENCES

1. Task Group, Air pollution and cancer. Risk assessment methodology and epidemiological evidence. Report of a Task Group. *Environ. Health Perspect.* 22: 1-12 (1978).
2. Stocks, P., and Campbell, J. M. Lung cancer death rates among nonsmokers and pipe and cigarette smokers—an evaluation in relation to air pollution by benzpyrene and other substances. *Brit. Med. J.* 2: 923-939 (1955).
3. Santodonato, J., Howard, P., and Basu, D. Health and ecological assessment of polynuclear aromatic hydrocarbons. *J. Environ. Pathol. Toxicol.* 5: 1-364 (1981).
4. Committee on Biologic Effects of Atmospheric Pollutants. Particulate Polycyclic Matter. Biological Effects of Atmospheric Pollutants. National Academy of Sciences, Washington, DC, 1972.
5. Carnow, B. W. The "urban factor" and lung cancer: cigarette smoking or air pollution? *Environ. Health Perspect.* 22: 17-21 (1978).
6. Teppo, L., and Pukkala, E. Keuhkosyövän yleistyminen Suomessa pysähtynyt (Lung cancer is no longer increasing in Finland.) *Duodecim* 96: 221-229 (1980).
7. Rimpelä, M. Aikuisväestön tupakointitavat Suomessa 1950-1970 -luvulla (Adult use of tobacco in Finland in the 1950s to 1970s). *Kansanterveystieteen julkaisuja M 40/78*. Kansanterveystieteen laitos, Tampere 1-65 (1978).
8. Levin, L., Morton, L., Haenszel, W., Carroll, B. E., Gerhardt, P., Handy, V. H., and Ingraham, S. C. Cancer incidence in urban and rural areas of New York State. *J. Natl. Cancer Inst.* 24: 1243-1257 (1960).
9. Blot, W. J., and Fraumeni, J. F., Jr. Original contributions. Geographic patterns of lung cancer: industrial correlations. *Am. J. Epidemiol.* 103: 539-550 (1976).
10. Teppo, L., Pukkala, E., Hakama, M., Hakulinen, T.,

- Herva, A., and Saxén, E. Way of life and cancer incidence in Finland. *Scand. J. Social Med. (Suppl.)* 19: 1-84 (1980).
11. Stocks, P. Recent epidemiological studies of lung cancer mortality, cigarette smoking, and air pollution with discussion of a new hypothesis of causation. *Brit. J. Cancer*, 20: 595-623 (1966).
 12. Häsänen, E., Karlsson, V., Leppämäki, E., and Juhala, M. Short communication benzene, toluene and xylene concentrations in car exhausts and in city air. *Atmos. Environ.* 15: 1755-1757 (1981).
 13. Hammond, E. C. Quantitative Relationship between Cigarette Smoking and Death Rates (Natl. Cancer Inst. Monographs, Vol. 28) Natl. Cancer Inst., Bethesda, 1968, pp. 3-8.
 14. Hietanen, E., and Hirn, J. Yhteenveto suomalaisten altistumisesta elimistölle vieraille aineille (Chemical exposures of Finnish population). *Ympäristö ja Terveys* 13(2-3): 109-144 (1982).
 15. Sauli, H. Ammatti ja kuolleisuus 1971-75 (Occupational mortality in 1971-75). Tilastokeskus, Helsinki Tutkimuksia No. 54: 1-158 (1979).
 16. Pukkala, E., Teppo, L., and Hakulinen, T. Keuhkosyöpäsairastavuus eri ammattialoilla Suomessa (Incidence of lung cancer by occupation in Finland). *Sosiaalilääket. Aikak. Lehti* 6: 363-371 (1979).
 17. Axelson, O., and Sundell, L. Mining, lung cancer and smoking. *Scand. J. Work Environ. Health* 4: 46-52 (1978).
 18. Hakala, E., Anttonen, H., and Yrjänheikki, E. Polycyclic aromatic hydrocarbons in the mine atmosphere. In: Program of 30. Nordiska Yrkeshygieniska Mötet. G. Wickström and B. Engström (Eds.), Institute of Occupational Health, Helsinki, 1981, pp. 179-181.
 19. Schimberg, R. W., Pfäffli, P., and Tossavainen, A. Polycyclic aromatic hydrocarbons in foundries. *J. Toxicol. Environ. Health* 6: 5-6 (1980).
 20. Skyttä, E., Schimberg, R., and Vainio, H. Mutagenic activity in foundry air. *Arch. Toxicol. (Suppl.)* 4: 68-72 (1980).
 21. Tola, S. Overview of Finnish epidemiologic studies on occupational cancer. *Scand. J. Work Environ. Health (Suppl.)* 4: 133-139 (1981).
 22. Koskela, R.-S., Hernberg, S., Kärävä, R., Järvinen, E., and Nurminen, M. A mortality study of foundry workers. *Scand. J. Work Environ. Health (Suppl.)* 1: 73-89 (1976).
 23. Tola, S., Koskela, R.-S., Hernberg, S., and Järvinen, E. Lung cancer mortality among iron foundry workers. *J. Occup. Med.* 21: 753-760 (1979).
 24. Vigliani, E. C., and Saita, G. Benzene and leukemia. *N. Engl. J. Med.* 271: 872-876 (1964).
 25. Fredga, K., Dävring, L., Sunner, M., Bengtsson, B. O., Elinder, C. G., Sigtryggsson, P., and Berlin, M. Kromosomförändringar hos arbetare exponerade för drivmedel och avgaser (Chromosome changes in workers exposed to motor fuels and their combustion residues). Institute of Environmental Health, Karolinska Institute, Stockholm; Institute of Environmental Health and Institute of Genetics, Lund 1979, Report 79 03 20.
 26. Nordenson, I., Sweins, A., Dahlgren, E., and Beckman, L. A study of chromosomal aberrations in miners exposed to diesel exhausts. *Scand. J. Work Environ. Health* 7: 14-17 (1981).
 27. Vainio, H., Sorsa, M., Rantanen, J., Hemminki, K., and Aitio, A. Biological monitoring in the identification of the cancer risk of individuals exposed to chemical carcinogens. *Scand. J. Work Environ. Health* 7: 241-251 (1981).